Original research

Clinical implications of gender and race in patients admitted with autoimmune hepatitis: updated analysis of US hospitals

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ABSTRACT

Background Autoimmune hepatitis (AIH) can result in end-stage liver disease that requires inpatient treatment of the hepatic complications. Given this phenomenon, it is important to analyse the impact of gender and race on the outcomes of patients who are admitted with AIH using a national hospital registry.

Methods The 2012–2017 National Inpatient Sample database was used to select patients with AIH, who were stratified using gender and race (Hispanics and blacks as cases and whites as reference). Propensity score matching was employed to match the controls with cases and compare mortality, length of stay and hepatic complications.

Results After matching, there were 4609 females and 4609 males, as well as 3688 blacks and 3173 Hispanics with equal numbers of whites, respectively. In multivariate analysis, females were less likely to develop complications, with lower rates of cirrhosis, ascites, variceal bleeding, hepatorenal syndrome, encephalopathy and acute liver failure (ALF); they also exhibited lower length of stay (adjusted OR, aOR 0.96 95% CI 0.94 to 0.97). When comparing races, blacks (compared with whites) had higher rates of ALF and hepatorenal syndrome related to ALF, but had lower rates of cirrhosis-related encephalopathy; in multivariate analysis, blacks had longer length of stay (aOR 1.071, 95% CI 1.050 to 1.092). Hispanics also exhibited higher rates of hepatic complications, including ascites, varices, variceal bleeding, spontaneous bacterial peritonitis and encephalopathy.

Conclusion Males and minorities are at a greater risk of developing hepatic complications and having increased hospital costs when admitted with AIH.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ While prior studies suggest a differential pattern of disease progression among autoimmune hepatitis (AIH) patients depending on gender and race, less is known about the gender and race-specified patterns of hepatic decompensation and liver failure that are observed in admitted AIH patients.

WHAT THIS STUDY ADDS

⇒ In this study, we stratify the admitted AIH population using gender and race in order to define the patterns of liver failure and hepatic decompensation among the susceptible cohorts. Furthermore, we use a propensity-score matched analysis in order to control for various medical confounders when assessing the relationships.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ By delineating the patterns of hepatic decompensation that are observed in gender and race-stratified strata of AIH patients, we are able to better understand disparities in outcomes observed in AIH cohorts, and furthermore improve the prognostication of risks in these vulnerable patients on their hospital admission.

INTRODUCTION

In patients with autoimmune hepatitis (AIH), failed immune tolerance leads to T-cell-mediated destruction of the hepatic parenchyma and stellate cellular production of matrices in the interstitial space.[1 2] These matrices culminate in fibrosis and lead to cirrhosis,[3 4] which can cause

complications such as portal hypertension, ascites, variceal bleeding, encephalopathy and coagulopathy,[5 6] affecting mortality and morbidity.[7–9] In conjunction with the natural course of untreated or advanced AIH, studies have investigated the roles of predisposing genetic and racial components in the prognostics of AIH-associated liver disease. These studies have noted signature differences in disease phenotypes stratified per race and genetic composition.[10–12] However, since the studies are primarily based on institutional data collection, further validation is required from a clinical perspective to understand differences in phenotype using race/gender while concurrently exploring the differences in hepatic and extrahepatic manifestations in hospital settings.

This study aims to evaluate the effects of race and gender in patients with AIH using national hospital data, specifically focusing on hepatic and extrahepatic comorbidities that result in hospital admission.

METHODS

Database

Funded by the Agency for Healthcare Research and Quality through the Healthcare Cost and Utilisation Project, the National Inpatient Sample (NIS) aggregates data compiled from statewide inpatient databases (SID) that comprise hospital claims data collected from predesignated states.[13 14] The database includes data from 2012 to 2017 sampled systematically across SID databases.[15–17] The discharge diagnoses are encoded using ICD 9 or ICD 10.[18 19] Variables were selected through a cross-referencing programme involving the General Equivalence Mappings base,[20 21] which converts between the ICD 9 and 10 systems.[22]

Weighted analysis

The formal weighing method as delineated by the NIS was used to delegate appropriate hospital-level strata and year information for the weighting analysis.[23 24] In addition to using the yearly estimates for each captured variable, trend analyses were performed for study variables and endpoints as stratified by predefined covariate terms. Best-fit regression analysis was performed to calculate the trend R^2 and p values.

Missing information

For the missing data, multiple imputations with chained equations were used to populate missing data. This method has been verified per literature to be an effective tool for representing missing data in administrative/large-database-driven studies.[25–27]

Comparative statistics

From NIS, the cohort of interest was found by isolating the in-hospital population with the cohort diagnoses. From this, exclusion criteria were applied to subset the final population of interest. Those under 18 years of age were excluded. The exposure variable was the diagnosis of AIH as defined using corresponding ICD terms. The endpoints included primary outcomes: mortality, length of stay and discharge disposition; secondary outcomes included ascites, varices, variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome and encephalopathy. Each secondary outcome was analysed as a composite variable and as pertaining to the main underlying liver complication: cirrhosis or acute liver failure (ALF). The cohort was further stratified by gender and race.

To minimise covariate confounding, propensity scores were derived for each case using subselected covariate terms. The covariates were fitted into a multivariate prediction model to derive the propensity scores, and terms included: diabetes, hyperlipidaemia, hypertension, chronic obstructive pulmonary disease (COPD), coronary artery disease, chronic kidney disease, congestive heart failure, coagulopathies and smoking. Furthermore, depending on the strata of interest (ie, gender or racial category), the non-used variable was inserted into the propensity score-generating model. Once the model was generated, the nearest neighbour mode was utilised to create 1:1 matches between the cases and controls. For gender comparisons, males were used as the reference group; for race comparisons, Whites were used as the reference group.

To generate univariate comparisons, the Jarque-Bera test was used to analyse variable parametricity.[28 29] χ^2 or Fisher's exact test was used for the analysis of nominal variables, and Student's t-test or Whitney-Mann U test was used for the analysis of non-nominal variables. To generate the multivariate analysis, the variable terms corresponding to hospital admission characteristics were imputed in the regression equations as covariates. P values ≤ 0.05 were designated statistical significance. Crude and adjusted ORs (aOR) with 95% CIs were quantified for nominal comparisons.

RESULTS

Patient selection

Figure 1 demonstrates the patient selection process. Patients with AIH were selected for this study and stratified either by gender or race. For the gender comparison cohort, there were a total of 21 787 patients including 17 178 females and 4609 males. After matching, there were 9218 patients, including 4609 female and 4609 male patients. For the race comparison cohort, there were a total of 14 926 white patients, 3688 black patients and 3173 Hispanic patients. After matching, 7376 patients were stratified into 3688 black patients and 3688 white patients, and 6346 patients were stratified into 3173 Hispanic patients and 3173 white patients for comparison.

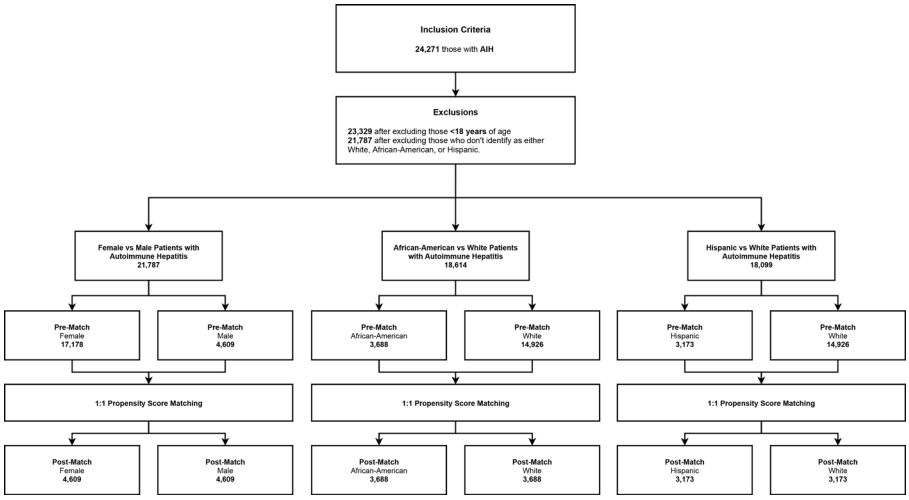

Figure 1 This figure shows the patient selection procedure of the study. AIH, autoimmune hepatitis.

Postmatch comparison of demographics and comorbidities

Table 1 compares prematch and postmatch demographics and medical comorbidities between female and male patients admitted with AIH. After matching, there were no differences in age and racial distribution. In terms of medical comorbidities, females persistently had lower rates of COPD (6.92% vs 8.03%, p=0.05) and congestive heart failure (8.53% vs 10.10%, p<0.01). Females also had lower rates of primary liver cancer (1.41% vs 2.26%, p<0.01), which includes hepatocellular carcinoma (1.30% vs 1.87%, p=0.04) and cholangiocarcinoma (0.11% vs 0.39%, p=0.01), alcoholic liver disease (2.02% vs 5.92%, p<0.001) and hepatitis B (0.09% vs 0.52%, p<0.001). Females also had a lower rate of postmatch malnutrition (9.16% vs 11.90%, p<0.001), which includes protein-calorie malnutrition, sarcopenia and cachexia.

Table 2 compares prematch and postmatch demographics and medical comorbidities between racial groups. Comparing black and white patients after matching, there were no significant differences in age, gender or comorbidities, except for greater rate of coronary artery disease (8.57% vs 6.94%, p=0.01) in blacks. There were fewer discrepancies in the rate of medical comorbidities postmatch. In terms of postmatch liver aetiologies, black patients had increased hepatitis B (0.73% vs 0.11%, p<0.001), increased hepatitis C (1.82% vs 0.60%, p<0.001) and decreased non-alcoholic fatty liver disease (2.58% vs 4.26%, p<0.001). Blacks were found to have a higher rate of malnutrition (10.90% vs 9.06%, p=0.01).

Comparing postmatch Hispanic and white patients, there were no significant differences in age, gender or comorbidities, except for greater rates of chronic kidney disease (15.10% vs 13.10%, p=0.03) and congestive heart failure (8.98% vs 7.50%, p=0.04) in Hispanic patients. Hispanics had greater rates of primary liver cancer (2.62% vs 1.32%, p<0.001), hepatocellular carcinoma (2.55% vs 1.17%, p<0.001), non-alcoholic fatty liver disease (5.26% vs 3.88%, p=0.01), hepatitis B (0.25% vs 0.03%, p=0.04) and hepatitis C (1.29% vs 0.54%, p=0.002) after matching.

Comparison of hospital outcomes and liver complications

Table 3 compares postmatch hospital outcomes between female and male patients. Female patients had a lower rate of mortality (3.10% vs 4.36%, p=0.002; OR 0.70, 95% CI 0.56 to 0.87)) than male patients but no difference in the length of stay. Females were less likely to undergo routine discharge. In terms of liver complications, female patients had a lower rate of cirrhosis (34.20% vs 41.60%, p<0.001), ALF (4.21% vs 5.25%, p=0.02), ascites (12.50% vs 17.30%, p<0.001; OR 0.68 95% CI 0.61 to 0.77), varices (11.80% vs 15.10%, p<0.001; OR 0.75, 95% CI 0.66 to 0.84), cirrhosis-related variceal bleeding (1.91% vs 2.65%, p=0.02; OR 0.72, 95% CI 0.54 to 0.94), cirrhosis-related spontaneous bacterial peritonitis (1.32% vs 2.78%, p<0.001; OR 0.47, 95% CI 0.35 to 0.64), cirrhosis-related hepatorenal syndrome (1.13% vs 2.10%, p<0.001; OR 0.53, 95% CI 0.38 to 0.75) and cirrhosis-related encephalopathy (8.16% vs 11.00%, p<0.001; OR 0.72, 95% CI 0.63 to 0.83). In the multivariate analysis, female patients had lower length of stay (p<0.001; aOR 0.96, 95% CI 0.94 to 0.97) and lower mortality (p=0.003; aOR 0.71, 95% CI 0.57 to 0.89). Figure 2 shows the multivariate analysis using gender as exposure and mortality as the primary endpoint.

Table 4 compares postmatch hospital outcomes between racial groups. In univariate comparison, black patients had a longer length of stay (6.4 vs 5.60 days, p<0.001) compared with white patients; there was no significant difference in mortality or disposition after discharge. In terms of liver complications, blacks had a greater rate of ALF (6.64% vs 4.37%, p<0.001), in the setting of a lower rate of encephalopathy (8.19% vs 9.76%, p=0.02). When further analysed by underlying

Table 1 Comparison of demographics and medical comorbidities in patients admitted with autoimmune hepatitis; male versus female

	Prematch comparison						Postmatch comparison					
	Female		Male			Univariate analysis	Female		Male			Univariate analysis
Demographics	n=17 178	78.85%	n=4609	21.15%	P value	OR (95% CI)	n=4609	50%	n=4609	50%	P value	OR (95% CI)
Age (years)	58.3	±17.60 years	54.1	±18.30 years	<0.001***		54	±18.10 years	54.1	±18.30 years	0.7	
Race					<0.001***						0.65	
White (%)	11 579	67.4	3347	72.6			3373	73.2	3347	72.6		
Black (%)	2981	17.4	707	15.3			675	14.6	707	15.3		
Hispanic (%)	2618	15.2	555	12			561	12.2	555	12		
Comorbidities												
Diabetes (%)	4678	27.2	1292	28	0.29	0.96 (0.89 to 1.03)	1283	27.8	1292	28	0.85	0.99 (0.90 to 1.08)
Hyperlipidaemia (%)	3750	21.8	994	21.6	0.72	1.02 (0.94 to 1.10)	968	21	994	21.6	0.52	0.97 (0.88 to 1.07)
Hypertension (%)	6397	37.2	1384	30	<0.001***	1.38 (1.29 to 1.48)	1380	29.9	1384	30	0.95	1 (0.91 to 1.09)
Chronic obstructive pulmonary disease (%)	1726	10	370	8.03	<0.001***	1.28 (1.14 to 1.44)	319	6.92	370	8.03	0.05*	0.85 (0.73 to 1.00)
Coronary artery disease (%)	1908	11.1	719	15.6	<0.001***	0.68 (0.62 to 0.74)	670	14.5	719	15.6	0.16	0.92 (0.82 to 1.03)
Chronic kidney disease (%)	2536	14.8	816	17.7	<0.001***	0.81 (0.74 to 0.88)	787	17.1	816	17.7	0.44	0.96 (0.86 to 1.07)
Congestive heart failure (%)	2018	11.7	467	10.1	0.002**	1.18 (1.06 to 1.31)	393	8.53	467	10.1	0.009**	0.83 (0.72 to 0.95)
Coagulopathies (%)	769	4.48	227	4.93	0.21	0.9 (0.78 to 1.05)	203	4.4	227	4.93	0.26	0.89 (0.73 to 1.08)
Smoking (%)	4264	24.8	1387	30.1	<0.001***	0.77 (0.71 to 0.82)	1365	29.6	1387	30.1	0.63	0.98 (0.89 to 1.07)
Liver aetiologies												
Primary liver cancer (%)	246	1.43	104	2.26	<0.001***	0.63 (0.50 to 0.79)	65	1.41	104	2.26	0.003**	0.62 (0.45 to 0.85)
Hepatocellular carcinoma (%)	225	1.31	86	1.87	0.006**	0.7 (0.54 to 0.90)	60	1.3	86	1.87	0.04*	0.69 (0.50 to 0.97)
Cholangiocarcinoma (%)	21	0.12	18	0.39	<0.001***	0.31 (0.17 to 0.59)	5	0.11	18	0.39	0.01*	0.28 (0.10 to 0.75)
Alcoholic liver diseases (%)	332	1.93	273	5.92	<0.001***	0.31 (0.27 to 0.37)	93	2.02	273	5.92	<0.001***	0.33 (0.26 to 0.42)
Hepatitis B (%)	39	0.23	24	0.52	0.002**	0.43 (0.26 to 0.72)	4	0.09	24	0.52	<0.001***	0.17 (0.04 to 0.48)
Hepatitis C (%)	171	1	73	1.58	0.001**	0.62 (0.47 to 0.82)	23	0.5	73	1.58	<0.001***	0.31 (0.19 to 0.50)
Non-alcoholic fatty liver disease (%)	710	4.13	199	4.32	0.61	0.96 (0.81 to 1.12)	175	3.8	199	4.32	0.22	0.87 (0.71 to 1.08)
Nutrition												

Continued

Table 1 Continued

Demographics	Prematch comparison						Postmatch comparison					
	Female		Male			Univariate analysis	Female		Male			Univariate analysis
	n=17 178	78.85%	n=4609	21.15%	P value	OR (95% CI)	n=4609	50%	n=4609	50%	P value	OR (95% CI)
Malnutrition (%)	1745	10.2	548	11.9	<0.001***	0.84 (0.76 to 0.93)	422	9.16	548	11.9	<0.001***	0.75 (0.65 to 0.85)

*P<0.05, **p<0.01, ***p<0.001.

liver complication, blacks had lower rates of cirrhosis-related encephalopathy (7.40% vs 9.33%, p=0.003; OR 0.78, 95% CI 0.66 to 0.92), while no difference was observed in ALF-related encephalopathy. Blacks also had greater rates of ALF-related hepatorenal syndrome (0.90% vs 0.43%, p=0.02; OR 2.07, 95% CI 1.14 to 3.77). In the multivariate analysis, blacks had a longer length of stay (p<0.001; aOR 1.071, 95% CI 1.050 to 1.092) but no difference in mortality compared with whites.

Hispanic patients had a longer length of stay (6.00 vs 5.68 days, p=0.033) and higher rate of routine discharge compared with white patients. There was no difference in mortality between Hispanic and white patients. In terms of liver complications, Hispanics had greater rates of cirrhosis (49.40% vs 37.30%, p<0.001), cirrhosis-related ascites (18.70% vs 14.40%, p<0.001, OR 1.37, 95% CI 1.20 to 1.56), cirrhosis-related varices (14.50% vs 9.74%, p<0.001; OR 1.57, 95% CI 1.35 to 1.83), cirrhosis-related variceal bleeding (3.72% vs 1.99%, p<0.001; OR 1.57, 95% CI 1.34 to 1.83), cirrhosis-related spontaneous bacterial peritonitis (2.74% vs 1.42%, p<0.001; OR 1.96, 95% CI 1.36 to 2.82) and cirrhosis-related encephalopathy (14.00% vs 10.80%, p<0.001; OR 1.35, 95% CI 1.16 to 1.57). There were no significant differences in ALF-related liver complications between the two cohorts. In the multivariate analysis, Hispanics were shown to have no difference in mortality or length of stay compared with white patients. Figure 3 represents the multivariate model using race as exposure and mortality as the primary endpoint.

Supplementary tables

Online supplemental table 1 shows the ICD codes used in the study. Online supplemental table 2 shows the comparison of socioeconomic and hospital characteristics stratified by gender and race. Online supplemental table 3 shows the prematch comparisons of clinical outcomes stratified by gender and race. Online supplemental tables 4–6 show the comparisons of clinical outcomes between males and females within each racial group (white, black and Hispanic). Online supplemental table 7 shows the comparison of clinical outcomes between blacks and Hispanics.

DISCUSSION

This study examines the effects of race and gender in patients with AIH using weighted analysis with propensity-matched comparisons. The results demonstrate that female patients have a lower tendency to experience hepatic complications, mortality and other AIH-related adverse events, including ascites, varices, variceal bleeding, spontaneous bacterial peritonitis, hepatorenal syndrome and encephalopathy in comparison to their male counterparts.

A nationwide cohort study in Denmark regarding AIH corroborates the current findings of a significantly

Table 2 Comparison of demographics and medical comorbidities in patients admitted with autoimmune hepatitis; stratified by race

	Black versus white prematch comparison						Black versus white postmatch comparison					
	Black		White			Univariate analysis	Black		White			Univariate analysis
Demographics	**n=3688**	**19.81%**	**n=14 926**	**80.19%**	**P value**	**OR (95% CI)**	**n=3688**	**50%**	**n=3688**	**50%**	**P value**	**OR (95% CI)**
Age (years)	49.5	±18.10 years	60.3	±17.10 years	<0.001***		49.5	±18.10 years	49.3	±18.50 years	0.65	
Female (%)	2981	80.8	11 579	77.6	<0.001***	1.22 (1.11 to 1.33)	2981	80.8	2970	80.5	0.77	1.02 (0.91 to 1.14)
Comorbidities												
Diabetes (%)	1074	29.1	3885	26	<0.001***	1.17 (1.08 to 1.26)	1074	29.1	1049	28.4	0.54	1.03 (0.93 to 1.14)
Hyperlipidaemia (%)	708	19.2	3500	23.4	<0.001***	0.78 (0.71 to 0.85)	708	19.2	645	17.5	0.06	1.12 (1.00 to 1.26)
Hypertension (%)	1366	37	5487	36.8	0.77	1.01 (0.94 to 1.09)	1366	37	1349	36.6	0.7	1.02 (0.93 to 1.12)
Chronic obstructive pulmonary disease (%)	271	7.35	1698	11.4	<0.001***	0.62 (0.54 to 0.71)	271	7.35	240	6.51	0.17	1.14 (0.95 to 1.36)
Coronary artery disease (%)	316	8.57	2065	13.8	<0.001***	0.58 (0.52 to 0.66)	316	8.57	256	6.94	0.01*	1.26 (1.06 to 1.49)
Chronic kidney disease (%)	703	19.1	2171	14.5	<0.001***	1.38 (1.26 to 1.52)	703	19.1	658	17.8	0.19	1.08 (0.96 to 1.22)
Congestive heart failure (%)	418	11.3	1782	11.9	0.32	0.94 (0.84 to 1.06)	418	11.3	376	10.2	0.12	1.13 (0.97 to 1.30)
Coagulopathies (%)	210	5.69	578	3.87	<0.001***	1.5 (1.27 to 1.76)	210	5.69	205	5.56	0.84	1.03 (0.84 to 1.25)
Smoking (%)	928	25.2	4195	28.1	<0.001***	0.86 (0.79 to 0.93)	928	25.2	883	23.9	0.23	1.07 (0.96 to 1.19)
Liver aetiologies												
Primary liver cancer (%)	53	1.44	214	1.43	1	1 (0.74 to 1.36)	53	1.44	39	1.06	0.17	1.36 (0.90 to 2.07)
Hepatocellular carcinoma (%)	46	1.25	184	1.23	1	1.01 (0.73 to 1.40)	46	1.25	34	0.92	0.22	1.36 (0.87 to 2.12)
Cholangiocarcinoma (%)	7	0.19	30	0.2	1	0.94 (0.41 to 2.15)	7	0.19	5	0.14	0.77	1.4 (0.44 to 4.42)
Alcoholic liver diseases (%)	87	2.36	415	2.78	0.17	0.84 (0.67 to 1.07)	87	2.36	106	2.87	0.19	0.82 (0.61 to 1.09)
Hepatitis B (%)	27	0.73	28	0.19	<0.001***	3.92 (2.31 to 6.67)	27	0.73	4	0.11	< 0.001†***	6.79 (2.36 to 26.73)
Hepatitis C (%)	67	1.82	136	0.91	<0.001***	2.01 (1.50 to 2.70)	67	1.82	22	0.6	< 0.001***	3.08 (1.90 to 5.00)
Non-alcoholic fatty liver disease (%)	95	2.58	647	4.33	<0.001***	0.58 (0.47 to 0.73)	95	2.58	157	4.26	< 0.001***	0.59 (0.46 to 0.77)
Nutrition												
Malnutrition (%)	401	10.9	1528	10.2	0.27	1.07 (0.95 to 1.20)	401	10.9	334	9.06	0.01*	1.23 (1.05 to 1.43)
Demographics	Hispanic versus white prematch comparison						Hispanic versus white postmatch comparison					
	Hispanic		White		P value	Univariate analysis	Hispanic		White		P value	Univariate analysis
	n=3173	17.53	n=14 926	82.47		OR 95% CI	n=3173	50	n=3173	50		OR 95% CI
Age (years)	52.9	±17.20	60.3	±17.10	<0.001***		52.9	±17.20	52.7	±17.60	0.74	
Female (%)	2618	82.5	11 579	77.6	<0.001***	1.36 (1.23 to 1.51)	2618	82.5	2625	82.7	0.84	0.98 (0.86 to 1.12)
Comorbidities												
Diabetes (%)	1011	31.9	3885	26	<0.001***	1.33 (1.22 to 1.44)	1011	31.9	943	29.7	0.07	1.11 (0.99 to 1.23)
Hyperlipidaemia (%)	536	16.9	3500	23.4	<0.001***	0.66 (0.60 to 0.73)	536	16.9	530	16.7	0.87	1.01 (0.89 to 1.16)
Hypertension (%)	928	29.2	5487	36.8	<0.001***	0.71 (0.65 to 0.77)	928	29.2	947	29.8	0.62	0.97 (0.87 to 1.08)

Continued

Table 2 Continued

	Black versus white prematch comparison						Black versus white postmatch comparison					
	Black		White			Univariate analysis	Black		White			Univariate analysis
Demographics	**n=3688**	**19.81%**	**n=14 926**	**80.19%**	**P value**	**OR (95% CI)**	**n=3688**	**50%**	**n=3688**	**50%**	**P value**	**OR (95% CI)**
Chronic obstructive pulmonary disease (%)	127	4	1698	11.4	<0.001***	0.32 (0.27 to 0.39)	127	4	98	3.09	0.06	1.31 (1.00 to 1.71)
Coronary artery disease (%)	246	7.75	2065	13.8	<0.001***	0.52 (0.46 to 0.60)	246	7.75	236	7.44	0.67	1.05 (0.87 to 1.26)
Chronic kidney disease (%)	478	15.1	2171	14.5	0.47	1.04 (0.94 to 1.16)	478	15.1	416	13.1	0.03*	1.18 (1.02 to 1.35)
Congestive heart failure (%)	285	8.98	1782	11.9	<0.001***	0.73 (0.64 to 0.83)	285	8.98	238	7.5	0.04*	1.22 (1.02 to 1.46)
Coagulopathies (%)	208	6.56	578	3.87	<0.001***	1.74 (1.48 to 2.05)	208	6.56	195	6.15	0.54	1.07 (0.88 to 1.31)
Smoking (%)	528	16.6	4195	28.1	<0.001***	0.51 (0.46 to 0.56)	528	16.6	508	16	0.52	1.05 (0.92 to 1.20)
Liver aetiologies												
Primary liver cancer (%)	83	2.62	214	1.43	<0.001***	1.85 (1.43 to 2.39)	83	2.62	42	1.32	< 0.001***	2 (1.38 to 2.91)
Hepatocellular carcinoma (%)	81	2.55	184	1.23	<0.001***	2.1 (1.61 to 2.73)	81	2.55	37	1.17	< 0.001***	2.22 (1.50 to 3.29)
Cholangiocarcinoma (%)	2	0.06	30	0.2	0.11	0.31 (0.04 to 1.24)	2	0.06	5	0.16	0.45†	0.4 (0.04 to 2.44)
Alcoholic liver diseases (%)	103	3.25	415	2.78	0.17	1.17 (0.94 to 1.46)	103	3.25	89	2.8	0.34	1.16 (0.87 to 1.55)
Hepatitis B (%)	8	0.25	28	0.19	0.6	1.34 (0.61 to 2.95)	8	0.25	1	0.03	0.04*†	8.02 (1.07 to 355.31)
Hepatitis C (%)	41	1.29	136	0.91	0.06	1.42 (1.00 to 2.02)	41	1.29	17	0.54	0.002**	2.43 (1.38 to 4.29)
Non-alcoholic fatty liver disease (%)	167	5.26	647	4.33	0.03*	1.23 (1.03 to 1.46)	167	5.26	123	3.88	0.01**	1.38 (1.09 to 1.75)
Nutrition												
Malnutrition (%)	364	11.5	1528	10.2	0.04*	1.14 (1.01 to 1.28)	364	11.5	295	9.3	0.005**	1.26 (1.08 to 1.49)

*P<0.05, **p<0.01, ***p<0.001.
†

Table 3 Postmatch comparison of hospital outcomes in patients admitted with autoimmune hepatitis; male versus female

	Female		Male			Univariate analysis	Multivariate analysis	
Hospital outcomes	**n=4609**	**50%**	**n=4609**	**50%**	**P value**	**OR (95% CI)**	**aOR (95% CI)**	**P value**
Mortality (%)	143	3.1	201	4.36	0.002**	0.7 (0.56 to 0.87)	0.71 (0.57 to 0.89)	0.003**
Length of stay (days)	5.57		5.9		0.77		0.96 (0.94 to 0.97)	<0.001†**
Hospitalisation cost ($)	52 413		62 658		<0.001***		0.86 (0.86 to 0.86)	<0.001†**
Disposition at discharge					<0.001***			
Routine (%)	3039	65.9	3047	66.1				
Short-term hospital (%)	155	3.36	203	4.4				
SNF or other facility (%)	574	12.5	487	10.6				
Home healthcare (%)	659	14.3	615	13.3				
Left AMA (%)	39	0.85	55	1.19				
Died (%)	143	3.1	201	4.36				
Unknown (%)	0	0	1	0.02				
Liver complications								
Acute liver failure (%)	194	4.21	242	5.25	0.02*	0.79 (0.65 to 0.96)		
Cirrhosis (%)	1578	34.2	1919	41.6	<0.001***	0.73 (0.67 to 0.79)		
Ascites (%)‡	577	12.5	799	17.3	<0.001***	0.68 (0.61 to 0.77)		
Cirrhosis related (%)	560	12.2	777	16.9	<0.001***	0.68 (0.61 to 0.77)		
ALF related (%)	46	1	82	1.78	0.002**	0.56 (0.39 to 0.80)		
Varices (%)‡	400	8.68	576	12.5	<0.001***	0.67 (0.58 to 0.76)		
Cirrhosis related (%)	398	8.64	568	12.3	<0.001***	0.67 (0.59 to 0.77)		
ALF related (%)	15	0.33	32	0.69	0.02*	0.47 (0.25 to 0.86)		
Variceal bleeding (%)‡	11	1.56	18	2.55	0.26	0.6 (0.28 to 1.29)		
Cirrhosis related (%)	88	1.91	122	2.65	0.02*	0.72 (0.54 to 0.94)		
ALF related (%)	1	0.02	6	0.13	0.12§	0.17 (0.00 to 1.37)		
Spontaneous bacterial peritonitis (%)‡	65	1.41	131	2.84	<0.001***	0.49 (0.36 to 0.66)		
Cirrhosis related (%)	61	1.32	128	2.78	<0.001***	0.47 (0.35 to 0.64)		
ALF related (%)	10	0.22	17	0.37	0.25	0.59 (0.27 to 1.28)		
Hepatorenal syndrome (%)‡	61	1.32	110	2.39	<0.001***	0.55 (0.40 to 0.75)		
Cirrhosis related (%)	52	1.13	97	2.1	<0.001***	0.53 (0.38 to 0.75)		
ALF related (%)	18	0.39	28	0.61	0.18	0.64 (0.35 to 1.16)		
Encephalopathy (%)‡	399	8.66	527	11.4	<0.001***	0.73 (0.64 to 0.84)		
Cirrhosis related (%)	376	8.16	506	11	<0.001***	0.72 (0.63 to 0.83)		
ALF related (%)	48	1.04	44	0.96	0.75	1.09 (0.72 to 1.65)		

*P<0.05, **p<0.01, ***p<0.001.
†Used Poisson regression analysis.
‡These variables include both cirrhosis and ALF-related events counting overlapping incidences.
§Fisher's exact test.
ALF, acute liver failure; AMA, against medical advice; aOR, adjusted OR; SNF, skilled nursing facility.

higher rate of AIH-related deaths in male subjects.[30 31] The higher risk of diagnosing AIH-related hepatocellular carcinoma in males may indicate that further gender-related comorbidities are playing a role in the mortality rate of male patients with AIH. While further evidence is required to explain the gender-specific differences in AIH outcomes, sex-related dissimilarities in immunogenetics, hypothalamic–pituitary–gonadal system, and sex hormones are postulated to interplay with autoimmune liver diseases.[32 33] These factors can influence disease activity and progression.[33 34] Additionally, physician biases in treatment plans may play a role in disease prognosis. For instance, female patients are generally more likely to be prescribed medications than male patients.[35] As evident in our current study, these biased treatment strategies may potentially attenuate the risk of liver failure and other fulminant diseases in female AIH patients. Furthermore, female patients are more likely to attend designated appointments,[36 37] which may also contribute to better disease control and reduce hepatic complications and liver failure.

When examining racial differences in AIH outcomes, we found that black and Hispanic patients suffer higher rates of hepatic complications, consistent with prior studies that suggest that minority populations with AIH experience more severe liver manifestations.[38–40] This phenomenon can also be explained by differences

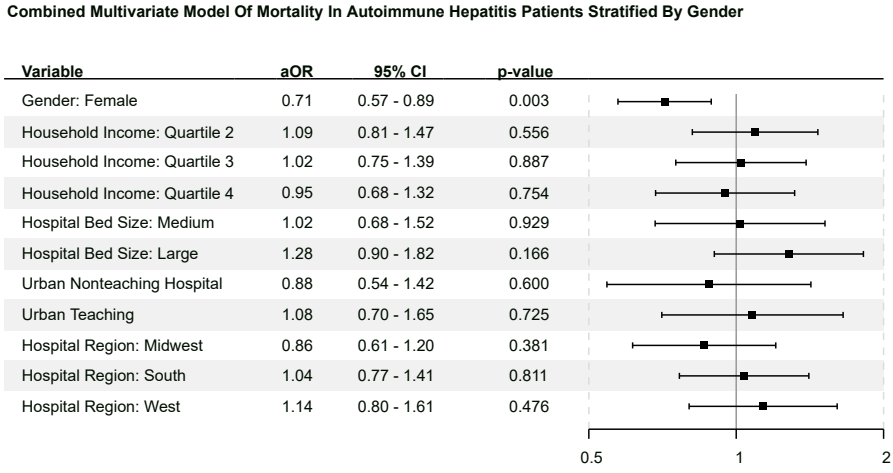

Figure 2 This figure is the multivariate forest plot representation using patient gender as exposure term and mortality as the endpoint.

in treatment response. Prior studies have demonstrated that minority populations experience variable treatment outcomes during pharmacological interventions due to racial differences in genetics and pharmacokinetics. Thus, physicians must consider these factors when making treatment plans to control AIH-induced hepatic inflammation.[41 42] As observed in our study, other studies have shown that other variables may be related to prolonged hospitalisation including severity of disease at time of presentation and challenges with placement at the time of discharge.[43]

Besides causes related to disease progression, socioeconomic factors and general medical accessibility may have also contributed to racial health disparities.[44] For instance, black patients experience limitations in insurance options and access to healthcare services compared with white patients. Furthermore, Hispanic patients and other minority racial groups may experience barriers due to health literacy and communication, which renders navigating through the hospital systems difficult.[45] Additionally, social and cultural biases may undermine effective patient–provider rapport, resulting in delays in diagnosis and treatment.[46–49] These inequalities in healthcare will likely lead to adverse outcomes in disease progression in both outpatient and inpatient settings.[44 50]

The current findings can be traced to symmetric disparities in outpatient approach and diagnostic workup. For instance, minority patients without access to primary care are more likely to have adverse AIH outcomes.[44] To overcome this, accessibility to medical care needs to be improved among minority populations via increasing AIH awareness and disease recognition, as well as implementing adjunctive measures (ie, health coordinators, case managers) to promote health literacy for vulnerable patients in navigating medical systems.[51] The burden of disease imposed on both patients and hospital systems can be curtailed by preventing the development of fulminant AIH disease through preventive or screening procedures. Given the differences in disease activity and progression from an inpatient perspective, diagnosis should not be delayed and there should be a lower threshold for starting immunosuppressive and other interventional therapies for AIH in minority patients. In particular, given the higher likelihood of dire complications observed in males and minority patients, there should be an earlier involvement of multidisciplinary services that can render various risk-appropriate levels of care.

CONCLUSION

Male patients experienced worse hospital outcomes, had higher rates of disease complications and higher hospital costs compared with female patients. Black and Hispanic patients experienced worse hospital outcomes, had higher rates of disease complications and higher hospital costs compared with white patients.

Contributors DL: roles: conceptualisation, data curation, formal analysis, investigation, methodology, supervision, validation, writing-original draft, writing-review and editing, manuscript submission; JK: roles: methodology, writing-original draft, visualisation, writing-review and editing; CK, JH, GHF, DJ, EAA and KC: roles: writing-original draft, investigation; NHU: roles: supervision, writing-review and editing. DUL acted as the guarantor.

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Data availability statement Data are available on reasonable request.

Table 4 Postmatch comparison of hospital outcomes in Patients admitted with autoimmune hepatitis; stratified by race

	Black		White			Univariate analysis	Multivariate analysis	
Hospital outcomes	**n=3688**	**50%**	**n=3688**	**50%**	**P value**	OR (95% **CI)**	**aOR (95% CI)**	**P value**
Mortality (%)	142	3.85	116	3.15%	0.11	1.23 (0.96 to 1.58)	1.2 (0.92 to 1.57)	0.17
Length of stay (days)	6.4	±7.41 days	5.6	±6.28 days	<0.001***		1.07 (1.05 to 1.09)	<0.001†**
Hospital charges ($)	62 220	±111 728	54 457	±101 707	<0.001***		1.06 (1.06 to 1.07)	<0.001†**
Disposition at discharge					0.51			
Routine (%)	2456	66.6	2486	67.4				
Short-term Hospital (%)	142	3.85	144	3.9				
SNF or other facility (%)	395	10.7	401	10.9				
Home healthcare (%)	514	13.9	492	13.3				
Left AMA (%)	39	1.06	48	1.3				
Died (%)	142	3.85	116	3.15				
Unknown (%)	0	0	1	0.03				
Liver complications								
Acute liver failure (%)	245	6.64	161	4.37	<0.001***	1.56 (1.27 to 1.91)		
Cirrhosis (%)	1338	36.3	1310	35.5	0.51	1.03 (0.94 to 1.14)		
Ascites (%)‡	470	12.7	490	13.3	0.51	0.95 (0.83 to 1.09)		
Cirrhosis related (%)	444	12	474	12.9	0.31	0.93 (0.81 to 1.07)		
ALF related (%)	61	1.65	51	1.38	0.39	1.2 (0.82 to 1.74)		
Varices (%)‡	348	9.44	315	8.54	0.19	1.12 (0.95 to 1.31)		
Cirrhosis related (%)	338	9.16	310	8.41	0.27	1.1 (0.94 to 1.29)		
ALF related (%)	28	0.76	19	0.52	0.24	1.48 (0.82 to 2.65)		
Variceal bleeding (%)‡	348	9.44	315	8.54	0.19	1.12 (0.95 to 1.31)		
Cirrhosis related (%)	57	1.55	59	1.6	0.93	0.97 (0.67 to 1.39)		
ALF related (%)	5	0.14	2	0.05	0.45§	2.5 (0.41 to 26.30)		
Spontaneous bacterial peritonitis (%)‡	67	1.82	69	1.87	0.93	0.97 (0.69 to 1.36)		
Cirrhosis related (%)	61	1.65	66	1.79	0.72	0.92 (0.65 to 1.31)		
ALF related (%)	10	0.27	8	0.22	0.81	1.25 (0.49 to 3.17)		
Hepatorenal syndrome (%)‡	75	2.03	67	1.82	0.55	1.12 (0.80 to 1.56)		
Cirrhosis related (%)	57	1.55	61	1.65	0.78	0.93 (0.65 to 1.34)		
ALF related (%)	33	0.9	16	0.43	0.02*	2.07 (1.14 to 3.77)		
Encephalopathy (%)‡	302	8.19	360	9.76	0.02*	0.82 (0.70 to 0.97)		
Cirrhosis related (%)	273	7.4	344	9.33	0.003**	0.78 (0.66 to 0.92)		
ALF related (%)	48	1.3	40	1.08	0.45	1.2 (0.79 to 1.83)		
Hospital outcomes	Hispanic		White		P value	Univariate analysis	Multivariate analysis	P value
	n=3173	50	n=3173	50		OR 95% CI	aOR 95% CI	
Mortality (%)	142	4.48	116	3.66%	0.11	1.23 (0.96 to 1.59)	0.99 (0.76 to 1.31)	0.96

Continued

Table 4 Continued

Hospital outcomes	Black n=3688	50%	White n=3688	50%	P value	Univariate analysis OR (95% CI)	Multivariate analysis aOR (95% CI)	P value
Length of stay (days)	6	±7.35 days	5.7	±6.75 days	0.03*		1 (0.98 to 1.02)	0.98†
Hospital charges ($)	72 233	± 123 440	58 070	±119 450	<0.001***		1.06 (1.06 to 1.06)	<0.001†**
Disposition at discharge					<0.001***			
Routine (%)	2210	69.7	2087	65.8				
Short-term hospital (%)	88	2.77	139	4.38				
SNF or other facility (%)	274	8.64	382	12				
Home healthcare (%)	430	13.6	423	13.3				
Left AMA (%)	28	0.88	26	0.82				
Died (%)	142	4.48	116	3.66				
Unknown (%)	1	0.03	0	0				
Liver complications								
Acute liver failure (%)	144	4.54	138	4.35	0.76	1.05 (0.82 to 1.33)		
Cirrhosis (%)	1569	49.4	1182	37.3	<0.001***	1.65 (1.49 to 1.82)		
Ascites (%)‡	604	19	469	14.8	<0.001***	1.36 (1.19 to 1.55)		
Cirrhosis related (%)	592	18.7	456	14.4	<0.001***	1.37 (1.20 to 1.56)		
ALF related (%)	52	1.64	39	1.23	0.21	1.34 (0.88 to 2.03)		
Varices (%)‡	463	14.6	312	9.83	<0.001***	1.57 (1.34 to 1.83)		
Cirrhosis related (%)	460	14.5	309	9.74	<0.001***	1.57 (1.35 to 1.83)		
ALF related (%)	16	0.5	13	0.41	0.71	1.23 (0.59 to 2.57)		
Variceal bleeding (%)‡	463	14.6	312	9.83	<0.001***	1.57 (1.34 to 1.83)		
Cirrhosis related (%)	118	3.72	63	1.99	<0.001***	1.91 (1.40 to 2.60)		
ALF related (%)	1	0.03	0	0	1§	Inf (0.03 to Inf)		
Spontaneous bacterial peritonitis (%)‡	89	2.8	47	1.48	<0.001***	1.92 (1.34 to 2.74)		
Cirrhosis related (%)	87	2.74	45	1.42	<0.001***	1.96 (1.36 to 2.82)		
ALF related (%)	7	0.22	7	0.22	1	1 (0.35 to 2.85)		
Hepatorenal syndrome (%)‡	80	2.52	60	1.89	0.1	1.34 (0.96 to 1.88)		
Cirrhosis related (%)	77	2.43	54	1.7	0.05	1.44 (1.01 to 2.04)		
ALF related (%)	12	0.38	14	0.44	0.84	0.86 (0.40 to 1.85)		
Encephalopathy (%)‡	455	14.3	364	11.5	<0.001***	1.29 (1.11 to 1.50)		
Cirrhosis related (%)	444	14	342	10.8	<0.001***	1.35 (1.16 to 1.57)		
ALF related (%)	29	0.91	40	1.26	0.23	0.72 (0.45 to 1.17)		

*P<0.05, **p<0.01, ***p<0.001.
†Used Poisson regression analysis.
‡These variables include both cirrhosis and ALF-related events counting overlapping incidences.
§Fisher's exact test.
ALF, acute liver failure; AMA, against medical advice; aOR, adjusted OR; SNF, skilled nursing facility.

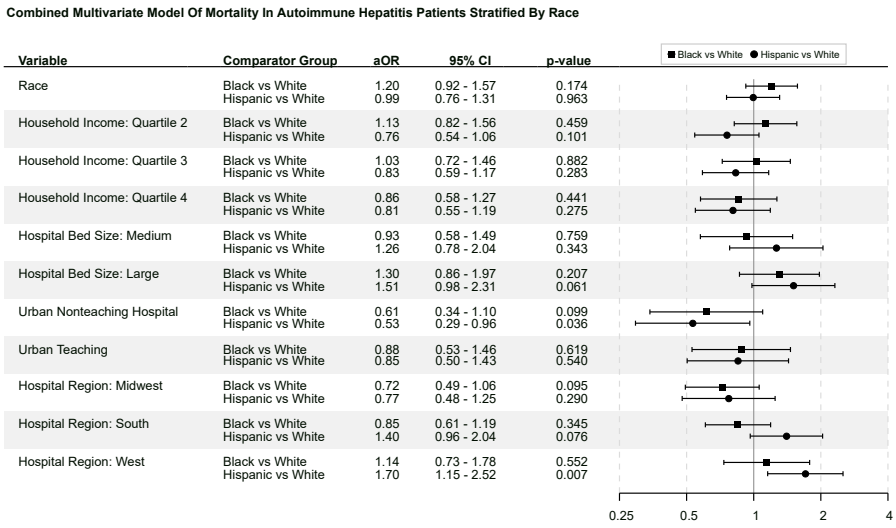

Figure 3 This figure shows the combinational multivariate forest plot using race as exposure and mortality as the endpoint.

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